rechallenged with an identical dose and schedule of IL-2 and γ-interferon without a recurrence of clinical symptoms and signs of pancreatitis or elevations of serum lipase or amylase levels (B.G.R., unpublished data, April 1989).

The acute onset of severe abdominal pain and ileus in patients treated with IL-2 is important because the possibility of spontaneous bowel perforation is a known complication.[5] Our experience, described herein, suggests the inclusion of acute pancreatitis in the differential diagnosis of acute abdominal pain in IL-2-treated patients. A careful physical examination and the appropriate laboratory and radiographic studies should be done to both diagnose acute pancreatitis and exclude spontaneous bowel perforation. Both of our patients responded to the cessation of IL-2 therapy and bowel rest as primary therapeutic maneuvers. We, therefore, recommend conservative therapy for the initial treatment of IL-2-associated pancreatitis. Whether pancreatitis will recur following the rechallenge of patients with IL-2-related pancreatitis with a further course of IL-2 therapy remains to be established.

REFERENCES

1. Rosenberg SA, Lotze MT, Muul LM, et al: A progress report on the treatment of 157 patients with advanced cancer using lymphokine-activated killer cells and interleukin-2 or high dose interleukin-2 alone. N Engl J Med 1987; 316:889-897

2. Rosenberg SA, Lotze MT, Muul LM, et al: Observations on the systemic administration of autologous lymphokine-activated killer cells and recombinant interleukin-2 to patients with metastatic cancer. N Engl J Med 1985; 313:1485-1492

3. Rosenberg SA, Lotze MT, Mule JJ: New approaches to the immunotherapy of cancer using interleukin-2. Ann Intern Med 1988; 108:853-864

4. Lotze MT, Matory YL, Rayner AA, et al: Clinical effects and toxicity of interleukin-2 in patients with cancer. Cancer 1986; 58:2764-2772

5. Schwartzentruber D, Lotze MT, Rosenberg SA: Colonic perforation: An unusual complication of therapy with high-dose interleukin 2. Cancer 1988; 62:2350-2353

6. Mallory A, Kern F Jr: Drug-induced pancreatitis: A critical review. Gastroenterology 1980; 78:813-820

7. Geokas MC, Baltaxe HA, Banks PA, et al: Acute pancreatitis. Ann Intern Med 1985; 103:86-100

8. Potts JR: Acute pancreatitis. Surg Clin North Am 1988; 68:281-299

9. Smith KA: Interleukin-2. Annu Rev Immunol 1984; 2:319-333

10. Gemlo BT, Palladino MA, Jaffe HS, et al: Circulating cytokines in patients with metastatic cancer treated with recombinant interleukin 2 and lymphokine-activated killer cells. Cancer Res 1988; 48:5864-5857

11. Estrin M, Huber SA: Coxsackievirus B3-induced myocarditis: Autoimmunity is L3T4+ T helper cell and IL-2 independent in BALB/c mice. Am J Pathol 1987; 127:335-341

Rhabdomyolysis and *Staphylococcus aureus* Septicemia in a Man With the Acquired Immunodeficiency Syndrome

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MYALGIA IS A COMMON COMPLAINT among patients with the acquired immunodeficiency syndrome (AIDS), particularly those taking zidovudine.[1] Rhabdomyolysis, a clinical syndrome of skeletal muscle injury, is an uncommon but important cause of myalgia and has been associated with staphylococcal septicemia in three patients without AIDS.[2-4] Infection with *Staphylococcus aureus* has been noted with increased frequency in patients with AIDS.[5-7] We report the case of a man with AIDS in whom diffuse myalgia developed and who was found to have *S aureus* septicemia with nonsuppurative rhabdomyolysis.

Report of a Case

The patient, a 43-year-old homosexual man, had two previous episodes of *Pneumocystis carinii* pneumonia that were successfully treated with regimens of trimethoprim-sulfamethoxazole, pentamidine isethionate, and dapsone-trimethoprim. Eight months before admission, therapy with zidovudine, 250 mg every four hours, was started; after five months the dose was decreased to 100 mg every four hours because of transient granulocytopenia. Other medical problems included depression, recurrent herpes genitalis and labialis, oral candidiasis, and dermatitis.

Four weeks before admission, he received influenza and pneumococcal immunizations. One and a half weeks before admission, he had the development of a nonproductive cough, malaise, and intermittent fever and chills. Four days later he began to have diffuse myalgia and noted difficulty initiating urine flow, with frequency and malodorous, dark urine. He also had painful sores on his tongue and soft palate and diarrhea. Three days before admission a visiting nurse noted a temperature of 39.6°C (103.3°F) and normal findings on a chest examination. The day before admission his muscle pains had so increased that he would not get out of bed. He also had dyspnea on exertion. On the morning of admission he was incontinent of stool and began to hallucinate. At the time of admission his medications included acyclovir sodium, 200 mg five times a day; lorazepam, 2 mg twice a day; and amitriptyline hydrochloride, 75 mg at bedtime.

On physical examination he was drowsy but arousable, with a temperature of 34.4°C (93.9°F), a heart rate of 125 beats per minute, a respiratory rate of 60 per minute, and a systolic blood pressure of 70 mm of mercury. He had acrocyanosis, left-sided rales, diffuse weakness, and pronounced muscle tenderness of the arms and legs. He had no rash, muscle swelling, or cardiac murmurs; his neck was supple and his abdomen showed no abnormalities.

Laboratory tests elicited the following values: a hemoglobin level of 112 grams per liter; leukocyte count of 1.0×10^9 per liter with 0.05 segmented neutrophils, 0.15 band forms, 0.13 metamyelocytes, 0.01 myelocytes, 0.34 lymphocytes, 0.29 monocytes, and 0.03 eosinophils with toxic granulations and Döhle's bodies; and a platelet count of 103×10^9 per liter. The prothrombin time was 13.2 seconds with a control of 11.7 seconds, and a partial thromboplastin time was 30.2 seconds with a control of 26.1. Arterial blood gas determinations made while the patient was breathing room

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ABBREVIATIONS USED IN TEXT
AIDS = acquired immunodeficiency syndrome
HIV = human immunodeficiency virus

air showed a pH of 7.39, a Po_2 of 113 mm of mercury, a Pco_2 of 21mm of mercury, with a carbon dioxide content of 14 mmol per liter and an anion gap of 19. A serum sodium level was 133 mmol per liter, potassium 4.4 mmol per liter, urea nitrogen 25 mmol per liter, creatinine 503.9 μmol per liter, glucose 8.7 mmol per liter, calcium 2.1 mmol per liter, phosphate 2.0 mmol per liter, aspartate aminotransferase 303 units per liter, albumin 26 grams per liter, and creatine kinase 11,700 units per liter. The urine was yellow and cloudy with a specific gravity of 1.014, pH 5.0, 1+ glucose, 2+ protein, moderate occult blood, moderate bacteria, rare red blood cells, and 6 to 10 white blood cells per high-power field with no casts. The urine was not examined for myoglobin. A cerebrospinal fluid analysis was unremarkable. A chest roentgenogram showed bilateral patchy infiltrates. An electrocardiogram showed sinus tachycardia with nonspecific ST-T wave changes.

Based on the initial diagnosis of sepsis and rhabdomyolysis, a regimen of cefazolin sodium, 1 gram; ampicillin, 2 grams; and ceftazidime, 2 grams, was given intravenously, and oxygen therapy by nasal cannula was started. He also received 5 liters of intravenous fluid with a urine output of 2.7 liters over 12 hours, but he remained hypotensive and complained of severe myalgia. A second laboratory examination showed the urea nitrogen level to be 23.9 mmol per liter, creatinine 389 μmol per liter, carbon dioxide content 9 mmol per liter, magnesium 0.91 mmol per liter, aspartate aminotransferase 628 units per liter, lactate dehydrogenase 111 units per liter, and creatine kinase 33,780 units per liter. His respirations ceased 12 hours after admission, and, in accordance with his previous request, he was not resuscitated.

Penicillinase-positive *Staphylococcus aureus,* sensitive to cefazolin, nafcillin, and gentamicin, grew from blood and urine (greater than 10^5 colonies per milliliter) and from postmortem cultures of specimens of lung and prostate. Viral cultures of prostate, brain, muscle, and lung were negative for influenza, cytomegalovirus, adenovirus, and enterovirus.

On postmortem gross examination there were pulmonary edema with bilateral pleural effusions, splenomegaly, left pyelonephritis, and prostatic abscesses. There was no muscle edema, hemorrhage, or degeneration, but the skeletal muscle appeared unusually dry. The heart was normal with coronary arteries free of atherosclerosis. Microscopic examination of the lung revealed scattered foci of *P carinii,* as well as cytomegalic cells. There were many renal abscesses. No myoglobin was seen. The prostate showed acute and chronic prostatitis with fibrosis, multiple abscesses, and many bacterial colonies. Cytomegalic cells, necrosis, and inflammation were seen in the central cortex of both adrenal glands. The bone marrow showed evidence of regeneration. There was widespread acute degeneration of skeletal muscle with loss of cross-striations and fragmentation of fibers. Neither inflammation nor organisms were seen (Figure 1). The final diagnosis was AIDS with *S aureus* septicemia, pyelonephritis, prostatitis, and Zenker's hyaline degeneration of muscle.

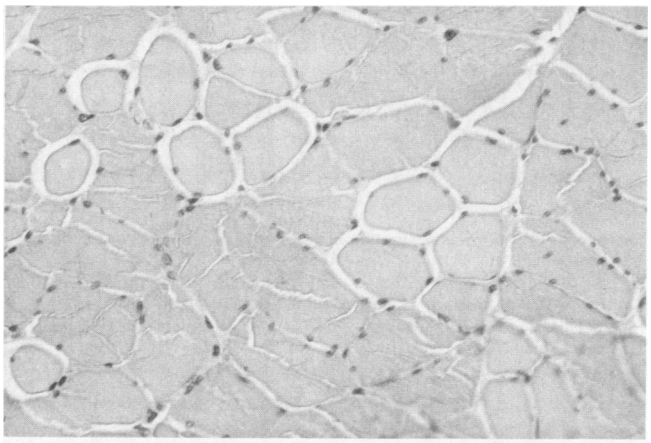

Figure 1.—A skeletal muscle biopsy shows fragmented fibers and hyalinized fibers lacking in cross-striations. No inflammation or organisms are present (hematoxylin and eosin; original magnification × 400).

Discussion

Myalgia is a common complaint among patients infected with the human immunodeficiency virus (HIV). In the phase II placebo-controlled trial of zidovudine in patients with AIDS and AIDS-related complex, myalgia was reported in 11% of patients who were taking zidovudine and 3% of those taking a placebo.[1]

Rhabdomyolysis is a clinical and laboratory syndrome resulting from skeletal muscle injury and the release of muscle cell contents into the plasma.[8] Evidence of rhabdomyolysis in this patient was the extreme elevation of creatine kinase levels and diffuse myalgia without apparent cardiac or brain injury. Acquired causes of rhabdomyolysis include excessive activity, direct muscle injury, ischemia, polymyositis, metabolic disorders, drugs, toxins, and infections. Rhabdomyolysis is an important cause of myalgia, though there is no evidence to suggest that it is more common in AIDS patients than in others.

There were several possible causes of rhabdomyolysis in our patient. While influenza infection is a recognized cause of rhabdomyolysis, influenza immunization is not, and it seems unlikely that influenza immunization was related to his subsequent illness.[8] His symptoms developed more than two weeks after immunization, and while convalescent-phase viral titers were not obtainable, viral cultures were negative for influenza and other viruses. Polymyositis has been described in cases of AIDS,[9] but in this case pathologic findings failed to support this diagnosis. Zidovudine therapy has been associated in four patients with a noninflammatory myopathy of an insidious onset.[10] In contrast, our patient had the sudden occurrence of myalgia that accompanied symptoms of acute infection. Profound hypotension may cause muscle ischemia with rhabdomyolysis,[8] and our patient was hypotensive in the last stages of his illness. His blood pressure was recorded as normal after the myalgia began, however.

We think staphylococcal bacteremia caused our patient's rhabdomyolysis and should be considered when AIDS patients present with this syndrome. Patients infected with HIV may be at an increased risk for staphylococcal infection. *Staphylococcus aureus* was reported by Whimby and coworkers in 32% of episodes of bacteremia among AIDS patients.[5] These cases occurred in association with *P carinii* pneumonia and pulmonary or cutaneous Kaposi's sarcoma.

Staphylococcus aureus accounted for 40% of cases of community-acquired bacteremia reported by Witt and colleagues[6]; all of these patients were intravenous drug users, however, making interpretation of causality difficult. Jacobsen and associates found that *S aureus* bacteremia was 20 times more likely to develop in patients with AIDS and AIDS-related complex than in age-matched controls with no risk factors for HIV infection.

The host defense against *S aureus* depends primarily on an intact phagocytic system and is compromised by granulocytopenia or by defective chemotaxis, phagocytosis, or intracellular killing. Cell-mediated immunity may modulate defenses against staphylococcal infection. Skin integrity is also important. In HIV-infected patients, a number of defects may contribute to an increased incidence of staphylococcal infection. Malnutrition, which is common in advanced HIV disease, may increase the susceptibility to infection including *Staphylococcus* species.[11] The high incidence of chronic, pruritic dermatologic disease in patients with HIV infection may provide a portal of entry for staphylococci that colonize the skin. Patients with Kaposi's sarcoma may have an increased incidence of staphylococcal infection if lesions erode or produce local lymphatic obstruction.[5] Granulocytopenia may be caused by zidovudine therapy[1] or complications of HIV infection.[12] Decreased chemotaxis of neutrophils[13] and monocytes[14] has also been observed in AIDS patients. In the absence of obvious malnutrition, dermatitis, granulocytopenia, or Kaposi's sarcoma, the latter mechanism remains the most plausible contributor. Staphylococci, however, are sufficiently virulent to invade apparently uncompromised patients.

There are several possible mechanisms by which *S aureus* infection might cause rhabdomyolysis. Skeletal muscle damage with *S aureus* usually occurs as a suppurative myositis: either as a large, localized abscess or many small abscesses. This condition usually occurs without sustained bacteremia and has been called pyomyositis.[15] Our patient had no evidence of staphylococcal invasion of muscle. Rhabdomyolysis has been seen in patients with toxic shock syndrome,[16-19] presumably due to effects of the exotoxin on muscle, but our patient did not appear to have this syndrome because he lacked the characteristic desquamating rash. We think that *S aureus* caused rhabdomyolysis in our patient through a nonsuppurative process, perhaps due to a different exotoxin.

Three previous cases of rhabdomyolysis associated with staphylococcal septicemia have been reported in patients without toxic shock. Adamski and co-workers described the case of a 15-year-old boy with nonsuppurative myositis and rhabdomyolysis.[2] The creatine kinase level was 33,400 units per liter. Gram-positive cocci were visible around some muscle fibers and *S aureus* grew on cultures. Lannigan and colleagues reported a fatal case of staphylococcal septicemia in a 70-year-old man.[3] A peak creatine kinase level was 83,000 units per liter, and a skeletal muscle biopsy specimen revealed multiple small areas of necrosis with microcolonies of *S aureus,* widespread muscle necrosis, and minimal inflammatory response. They hypothesized that a toxin plays a role in rhabdomyolysis. Saitoh and associates described endocarditis in a 20-year-old man who presented with fever, rash, generalized myalgia, and muscle weakness.[4] Blood and urine cultures were positive for *S aureus,* and a peak creatine kinase level was 990 units per liter. A muscle biopsy specimen showed focal necrosis and mild inflammation without bacterial invasion or suppuration. Saul and co-workers reported an additional case of a 13-year-old girl with rhabdomyolysis with a creatine kinase level of 59,729 units per liter and nonoliguric renal failure, who did not have toxic shock syndrome or bacteremia, but who had *Staphylococcus* organisms isolated from the urine.[20]

Our case differed from those previously reported in that it involved a patient with AIDS, and neither rash nor bacterial invasion of muscle was seen. This also appears to be the first case of Zenker's hyaline degeneration in association with staphylococcal infection, although the case reported by Saitoh and associates may have been another example. Nonsuppurative muscle damage from staphylococcal infection may be more likely to develop in AIDS patients, though this has not been studied.

Zenker first described in 1859 and 1862 the waxy or hyaline degeneration of skeletal muscle in 120 patients who died of typhoid fever.[21(p1580),22] Zenker's hyaline degeneration may occur in any muscle but is most common in the large muscles of the trunk, pelvis, and shoulder girdle. The muscle is grossly very pale. On microscopic examination, there is cloudy swelling of the fibers with a loss of striations and fragmentation into horizontal segments. Sarcolemmal nuclei are slightly swollen and may proliferate. After the first week, highly refractile, eosinophilic, cylindric masses may be seen within the muscle fibers. Hyaline segments are usually interspersed with healthy fibers. Phagocytic cells appear between fibers, but inflammatory cells are never prominent. The proposed mechanism of damage is either a microbial toxin or metabolic disorder in the muscle fibers induced by the infection. In addition to typhoid fever, a high incidence of Zenker's hyaline degeneration was also reported in smallpox. Because these associated diseases are rare in industrialized nations, Zenker's degeneration is an infrequent pathologic finding. It has been described less often in patients with other infectious diseases, including influenza, pneumonia, scarlet fever, diphtheria, tuberculosis, tetanus, cholera, hepatitis A, yellow fever, streptococcal septicemia, and noninfectious diseases such as advanced malignancy and cirrhosis.[22]

REFERENCES

1. Richman DD, Fischl MA, Grieco MH, et al: The toxicity of azidothymidine (AZT) in the treatment of patients with AIDS and AIDS-related complex: A double-blind, placebo-controlled trial. N Engl J Med 1987; 317:192-197

2. Adamski GB, Garin EH, Ballinger WE, et al: Generalized nonsuppurative myositis with staphylococcal septicemia. J Pediatr 1980; 96:694-697

3. Lannigan R, Austin TW, Vestrup J: Myositis and rhabdomyolysis due to *Staphylococcus aureus* septicemia. J Infect Dis 1984; 150:784

4. Saitoh H, Yoshii F, Ishihara T, et al: Rhabdomyolysis associated with staphylococcal septicemia. Rinsho Shinkeigaku 1986; 26:693-697

5. Whimby E, Gold JWM, Polsky B, et al: Bacteremia and fungemia in patients with the acquired immunodeficiency syndrome. Ann Intern Med 1986; 104:511-514

6. Witt DJ, Craven DE, McCabe WR: Bacterial infections in adult patients with the acquired immune deficiency syndrome (AIDS) and AIDS-related complex. Am J Med 1987; 82:900-906

7. Jacobsen MA, Gellerman H, Chambers H: *Staphylococcus aureus* bacteremia and recurrent staphylococcal infection in patients with AIDS and ARC (Abstr). 4th International Conference on AIDS, Stockholm, 1988, p 399

8. Gabow PA, Kaehny WD, Kelleher SP: The spectrum of rhabdomyolysis. Medicine (Baltimore) 1982; 61:141-152

9. Dalakas MC, Pezeshkpour GH, Gravell M, et al: Polymyositis associated with AIDS retrovirus. JAMA 1986; 256:2381-2383

10. Bessen LJ, Greene JB, Louie E, et al: Severe polymyositis-like syndrome associated with zidovudine therapy of AIDS and ARC (Letter). N Engl J Med 1988; 318:708

11. Keusch GT: Malnutrition and infection, *In* Braude A (Ed): Infectious Disease and Medical Microbiology. Philadelphia, WB Saunders, 1986, pp 709-714

12. Lane CH, Fauci AS: Immunologic abnormalities in the acquired immunodeficiency syndrome. Annu Rev Immunol 1985; 3:477-500

13. Nielsen H, Kharazmi A, Faber V: Blood monocyte and neutrophil functions in the acquired immune deficiency syndrome. Scand J Immunol 1986; 24:291-296

14. Prince HE, Moody DJ, Shubin BI, et al: Defective monocyte function in acquired immune deficiency syndrome (AIDS)—Evidence from monocyte-dependent T-cell proliferative system. J Clin Immunol 1985; 5:21-25

15. Armstrong JH: Tropical pyomyositis and myoglobinuria. Arch Intern Med 1978; 138:1145-1146

16. McKenna UG, Meadows JA, Brewere NS, et al: Toxic shock syndrome, a newly recognized disease entity—Report of 11 cases. Mayo Clin Proc 1980; 55:663-672

17. Chesney RW, Chesney PJ, Davis JP, et al: Renal manifestations of the staphylococcal toxic-shock syndrome. Am J Med 1981; 71:583-588

18. Scobie BA: Staphylococcal toxic shock: Two fulminant cases with recovery. NZ Med J 1982; 95:145-147

19. Bachhuber R, Parker RA, Bennet WM: Acute renal failure in toxic shock syndrome owing to rhabdomyolysis. Ann Clin Lab Sci 1983; 13:25-26

20. Saul RA, Vernon M, Roe C, et al: Rhabdomyolysis in a patient with nonoliguric renal failure: Similarities to the toxic-shock syndrome. South Med J 1980; 73:261-263

21. Kaufmann E: Lehrbuch der speziellen pathologischen Anatomie, Vol 2. Berlin, De Gruyter, 1922, p 1580

22. Kakulas BA: Diseases of Muscle. Philadelphia, Harper & Row, 1985, pp 590-594

Capgras's Syndrome Associated With Sensory Loss

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THE CAPGRAS SYNDROME is the delusion that a known person or object has been replaced by a virtually identical duplicate. Reported formally first by Capgras and Réboul-Lachaux as the delusion of doubles ("l'illusion des sosies"), more than 300 cases have been recorded in the English and French literature. The delusion has been found in cases of both idiopathic and secondary ("organic") psychoses.[1] It and several variants form the delusion of substitution[2] or the delusional misidentification syndromes.[3] While sensory loss is frequently cited as one initiator of hallucinations and delusions, a persistent circumscribed delusion of the Capgras type has not been reported in such cases, aside from that of a profoundly deaf man with a progressive dementia.[4]

Report of Cases

Case 1

The patient, a 75-year-old right-handed woman, believed that her spouse of 25 years had been replaced by another man; there has been some gradual accommodation to the "new man's" presence over a two-year period since the symptoms began. Although at least one other object (a bed) had been replaced, she did not think that her maid, natural and stepchildren, or friends had been affected. She believed her house was not the same one she left before being admitted to a hospital, describing it as being larger, having a different layout, and with stairs in places that did not exist previously. She was convinced that her original house and those of neighbors were really one block away and that she could "see" them when she walked by.

The patient had been healthy until four years before when giant cell arteritis developed. Over a two-week period she went blind in the right eye and had severe visual impairment in the left. On high doses of corticosteroids, *Aspergillus* species pneumonia developed from a dormant non-acid-fast pulmonary infection, and the left eye became infected, leading to complete blindness.

About 20 months after her illness began, she was not using any medication and was mildly to moderately depressed, with a sense of helplessness, weight loss, and apathy, but no suicidal ideation; there was no disorder of thought form. Attention, language, calculations, visual-spatial ability, and general fund of knowledge were intact; short-term memory only was mildly impaired. The cranial nerves were normal aside from absent light perception; the results of motor, cerebellar, and sensory examinations were normal. The electroencephalogram showed mild abnormalities with a posterior dominant rhythm of 7 Hz and generalized slowing of background frequencies; a computed tomographic scan showed mild generalized cerebral atrophy and slight enlargement of the ventricles, consistent with her age, but no evidence of a focal lesion. There was no change in her state despite a brief trial of antidepressants, although the depression has remitted somewhat with time.

Case 2

The patient, a 66-year-old widow, claimed to have last seen her daughter 18 months before evaluation, but reported hearing her voice intermittently, telling her how she was being tormented by her captors. She believed that her daughter had been hidden in a secret chamber next door and that "a woman" working at city hall pretended to be her daughter. The patient was terrified because her granddaughter had betrayed her own mother by living with the "imposter."

Her belief that her daughter had been replaced began 15 years earlier after surgical treatment of an ear infection eliminated her hearing in both ears; her daughter's voice was heard only on the right and became louder and more persistent after she removed her hearing aid on that side. No other delusional or affective symptoms were ascertainable. The patient had had insulin-dependent diabetes mellitus since age 30, as well as coronary artery disease and peripheral neuropathy. Although anxious and vociferous in her complaints, she had no cognitive impairment noted on examination and there was no history of social deterioration. She did not agree to further assessment or treatment and was released from hospital.

Discussion

Both patients demonstrated the Capgras syndrome, which appeared some time after a severe sensory loss, accompanied by hallucinations in the affected modality. In patient 1, the delusion persisted even though her medical and psychological state, aside from the residual blindness, had essentially returned to normal; the mild dysrhythmia has been found in some other cases of the syndrome. She had a co-existent symptom of reduplicative paramnesia. In the second patient, the long-term nature and lack of social deterioration suggested that her delusion was circumscribed, so that a diagnosis of late-onset paraphrenia could be considered. The

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